Amendments to the Claims

This listing of claims will replace all prior versions and listing of claims in the application:

CLAIMS

- 1-4. (Canceled)
- 5. (Currently amended) Animal model according to Claim [[3]] 16, characterized in that the mutation in the murine presentil 1 gene comprising a M146L mutation PS1 gene is replaced by or is in addition to a murine presentil 1 gene mutation is selected from the group consisting of M146L, A246E, C410Y, H163R, L286V and L235P mutations, taken on their own or in combination.
- 6. (Canceled)
- 7. (Currently amended) Animal model according to Claim 16, characterized in that the mitochondrial dysfunction is an alteration, a modification, an overexpression or an inhibition of the expression of the mitochondrial proteins.
- 8. (Original) Animal model according to Claim 7, characterized in that the proteins are intramitochondrial proteins.
- 9. (Original) Model according to Claim 8, characterized in that the proteins are the Bax and/or cytochrome c proteins.
- 10. (Currently amended) A method for identifying compounds which can be used for treating neurodegenerative diseases that modulate the amyloid plaques, neuronal loss or mitochondrial dysfunction of the non-human transgenic animal model of claim 16 comprising exposing said compounds to the animal model of any one of claims 1 to 9.

11 - 15. (Canceled)

16. (Currently amended) Non-human transgenic animal model that exhibits amyloid plaques, neuronal loss and mitochondrial dysfunction, said model comprising:

a nucleic acid sequence encoding a mutation in the murine presenilin 1 protein gene comprising a M146L mutation; and

a nucleic acid sequence encoding mutations in the human β -amyloid peptide protein precursor comprising the Swedish, Dutch and London mutations.